Computed Tomographic Reversal Sign in an Adult with Methanol Intoxication

Metanol İntoksikasyonlu Yetişkin Hastada Bilgisayarlı Tomografik Tersine Dönme İşareti

Metanol İntoksikasyonunda BT Tersine Dönme İşareti / CT Reversal Sign in Methanol Intoxication

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Özet

Bilgisayarlı tomografik (BT) tersine dönme işareti difüz anoksik-iskemik beyin hasarı ile birlikte olup hemen daima çocuklarda görülür. Literatürde sadece menenjitli yetişkin bir hastada bildirilmiştir. Metil alkol intoksikasyonu genellikle iki taraflı putaminal nekroz ile birliktedir. Metil alkol intoksikasyonunda BT tersine dönme işareti daha önce literatürde tanımlanmamıştır. Bu yazıda metal alkol intoksikasyonlu yetişkin bir hasta ve BT tersine dönme işareti sunulmaktadır.

Anahtar Kelimeler

Metanol İntoksikasyonu; BT Tersine Dönme İşareti; İskemik Beyin Hasarı

Abstract

The computed tomographic (CT) reversal sign is associated with diffuse, anoxic/ischemic cerebral injury, and is seen almost exclusively in children, and only one adult patient with meningitis exists in the literature with this sign. On the other hand, methyl alcohol intoxication is usually associated with bilateral putaminal necrosis, and no CT reversal sign has been observed before in this condition. This report presents an adult patient with methyl alcohol intoxication, and the CT reversal sign.

Keywords

Methanol Intoxication; CT Reversal Sign; Ischemic Cerebral Injury

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Introduction

The reversal sign, a striking computed tomographic (CT) finding, represents a diffuse, anoxic/ischemic cerebral injury. The CT reversal sign appears as decreased density of the white and gray matter with relatively increased density of the thalami, brainstem, and cerebellum [1-4]. The CT reversal sign is very rare in adults, and there is only one report of an adult with bacterial meningitis [5]. On the other hand, methyl alcohol intoxication is usually associated with bilateral putaminal necrosis, and hemorrhage and edema in the brain, and no CT reversal sign has been observed before [6-9]. In this communication, an adult patient is reported revealing the CT reversal sign due to methanol intoxication.

Case Report

A 57-year-old man, who reportedly ingested a large amount of alcohol starting 12 hours before, was admitted because of visual impairment, unconsciousness and apnea attacks. His mental status deteriorated until he became comatose. He had metabolic acidosis, and was intubated. Laboratory findings were as follows: Arterial blood gas investigation showed a low pH of 7.12 (normal pH 7.35–7.45) and HCO3 was 12 µmol/L (normal 22–26 µmol/L) causing severe metabolic acidosis. Toxicology screening revealed a methanol level of 30 mg/dl. The oculocephalic reflex was negative as were the light and corneal reflexes. Motor response to pain stimulation was negative in all extremities. Signs of pyramidal involvement were positive bilaterally. He was treated with bicarbonate and ethanol. Dexamethasone 16 mg/day parenterally was given to control the edema. The patient remained comatose and died 3 days after admission. On admission, a CT scan revealed an apparent hyperintensity of bilateral thalamus, brainstem, and cerebellum associated with hypointensity of the white and gray matter. There was no evidence of transtentorial herniation (Figures.1 A-C).

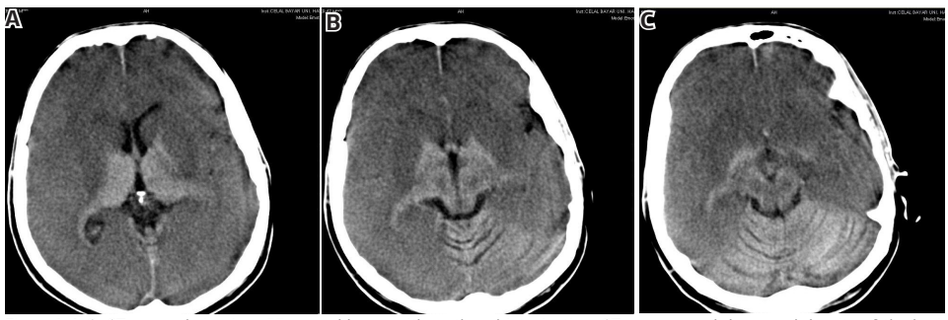

Figure.1 A-C. CT reversal sign in a 57-year-old man with methanol intoxication. CT scans reveal decreased density of the brain parenchyma with relatively increased density of the thalami (A), brainstem (B), and cerebellum (C).

Discussion

After methanol intake to the level toxic to the particular individual, there usually is a latent period of approximately 24 hours before the symptoms begin. This latent period may range between 1 and 72 hours, after which metabolic acidosis and clinical findings become apparent. The most characteristic clinical finding in metanol intoxication is visual impairment or cortical blindness. CT and magnetic resonance imaging (MRI) are highly useful modalities for demonstrations of brain damage where basal ganglia frequently are involved. Previous imaging studies have shown bilateral putaminal necrosis to be a characteristic feature of methanol intoxication [6,7,9].

Various studies also have defined other cerebral findings in methanol intoxication, including hemorrhage, edema and atrophy [6-9]. Never has been mentioned about the CT reversal sign in this condition. With respect to our patient, although bilateral putaminal necrosis is typical in methanol intoxication, there was no putaminal necrosis.

CT features of the reversal sign have been described as a diffusely decreased density of cerebral cortical gray and white matter with a decreased or lost gray/white matter interface, or reversal of the gray/white matter densities, and relatively increased density of the thalami, brainstem, and cerebellum. The CT reversal sign has usually been described in children for anoxic/ischemic states due to birth asphyxia, drowning, status epilepticus, and meningitis or degenerative encephalitis [1-4]. It is very rare in adults, and there is only one report of the CT reversal sign in an adult with bacterial meningitis [5]. Since the CT density of the thalami and brainstem appear normal (or relatively increased) the condition has also been referred to as the 'central structure preservation sign'. The pathogenesis of central structure preservation is not known. There have been two main explanations of the CT reversal sign [1-5]. First, a mechanical factor was suggested that the preservation was due to transtentorial herniation during acutely edematous state of the brain, and that locally reduced tissue pressure causes increased blood flow, and the improved tissue perfusion might then delay prevent tissue necrosis of the central region. The second explanation of the CT reversal sign was postischemic increased capillary proliferation in the central regions. The reversal sign is associated with a poor prognosis [1,2,5]. One-third of patients demonstrating this sign on CT scans will die. The remainders suffer severe permanent brain damage, later developing diffuse atrophy and cystic encephalomalacia [2, 5].

In our 57-year-old patient with methanol intoxication it seems difficult to explain the occurrence of CT reversal sign as this sign has exclusively been detected before in anoxic/ischemic states of children, with the exception of one adult case with meningitis. Since there was no imaging evidence of transtentorial herniation in our patient an increased capillary proliferation in the thalami and brainstem may be considered as the mechanism of the development of the CT reversal sign.

In conclusion, the reversal sign is a characteristic finding of the ischemic state of the adult brain in CT, and it demonstrates irreversible brain damage and carries a poor prognosis.

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